Anaesthesia
Journal of the Association of Anaesthetists of Great Britain and Ireland

Anaesthesia, 2009, **64**, pages 378–382 doi:10.1111/j.1365-2044.2008.05793.x

Comparison of central venous oxygen saturation and mixed venous oxygen saturation during liver transplantation

A. El Masry,[1] A. M. Mukhtar,[1] A. M. El Sherbeny,[1] M. Fathy[2] and M. El-Meteini[3]

1 Lecturer in Anaesthesia, Faculty of Medicine, Cairo University, Giza, Egypt
2 Assistant Professor and 3 Professor, Department of General Surgery, Ain-Shams University, Cairo, Egypt

Summary

Central venous catheterisation is commonly performed during major surgery and intensive care, and it would be useful if central venous oxygen saturation could function as a surrogate for mixed venous oxygen saturation. We studied 50 patients undergoing living related liver transplantation. Blood samples were taken simultaneously from central venous and pulmonary artery catheters at nine time points during the pre-anhepatic, anhepatic, and postanhepatic phases. Four hundred and fifty sets of measurement were obtained. There was a good correlation between central venous oxygen saturation and mixed venous oxygen saturation. The mean (SD) difference (95% limit of agreement) was lowest at the first time point (1.06 (0.65)%, −1.94% to 2.7%) and then increased throughout the study but remained acceptable. The change in mixed venous oxygen and central venous oxygen saturations occurred mostly in parallel and as a result changes in mixed venous oxygen saturation were reflected adequately in the change in central venous oxygen saturation. The correlation between mixed venous oxygen saturation and cardiac output was poor.

Correspondence to: Dr Ashraf El Masry
E-mail: aaaelmasry@yahoo.com
Accepted: 18 October 2008

True mixed venous blood is derived from a pool of venous blood entering the pulmonary artery via the great veins in the chest. It contains blood which has traversed all systemic capillary beds capable of extracting oxygen, and is thoroughly mixed by the right ventricle. It has been suggested that the most suitable sites with respect to complete mixing are the right ventricular outflow tract and pulmonary artery [1, 2]. Mixed venous oxygen saturation has been shown to be a surrogate for the balance between systemic oxygen delivery and consumption during treatment of critically ill patients [3]. Maintenance and monitoring of tissue oxygenation is one of the most important goals in the care of critically ill or unstable patients. Measurement of mixed venous oxygen saturation (Svo_2) requires placement of a pulmonary artery catheter, which may not be feasible in all patients. However, central venous access can be easily and safely obtained in both ICU and non-ICU settings, which makes central venous oxygen saturation ($Scvo_2$) monitoring a convenient surrogate for Svo_2. There has been considerable debate regarding whether $Scvo_2$ is a satisfactory substitute for Svo_2, particularly above 65% [4–6]. Although the absolute values of $Scvo_2$ and Svo_2 differ, previous studies have shown close tracking of the two measurements across a wide range of haemodynamic conditions [7].

Profound haemodynamic and metabolic changes occur during adult orthotropic liver transplantation (OLT) [5, 6]. During the dissection phase the disturbance is mainly due to bleeding and hypovolemia [8], while in the anhepatic phase there may be a 50% reduction of venous return with clamping of inferior vena cava [9]. Reperfusion of the graft during the neo-hepatic stage carries the risk of systemic hypotension (postreperfusion syndrome) [10]. The relationship between Svo_2 and $Scvo_2$ has not assessed in such conditions where haemodynamic changes occur over a short period of time depending on the surgical phase.

The aim of our study was to compare Svo_2 vs $Scvo_2$ in the various haemodynamic conditions that occur during the three phases of orthotropic liver transplantation. We prospectively studied the oxygen saturation of blood samples simultaneously taken from superior vena cava and pulmonary artery catheter during the pre-anhepatic, anhepatic, and postanhepatic phases of living related liver transplantation.

© 2009 The Authors
Journal compilation © 2009 The Association of Anaesthetists of Great Britain and Ireland

Methods

After approval of the local Ethics Committee and obtaining written informed consent, the study was designed to include 50 ASA III or IV patients with end-stage liver disease, scheduled for orthotropic liver transplantation between 2005 and 2006. Induction of anaesthesia was performed using propofol, fentanyl and atracurium. Anaesthesia was maintained with sevoflurane adjusted between 1–2% in an air/oxygen mix, fentanyl infusion at 1–2 $\mu g.kg^{-1}.h^{-1}$ and atracurium infusion at 0.5 $mg.kg^{-1}.h^{-1}$. Mechanical ventilation was provided using a tidal volume of 8–10 $ml.kg^{-1}$ with the respiratory rate adjusted to maintain the $PaCO_2$ between 4–4.6 kPa. All patients were monitored for temperature, non-invasive and invasive arterial blood pressure, five lead ECG, peripheral oxygen saturation, end-tidal carbon dioxide tension, hourly urinary output, and central venous pressure (CVP). A 7-Fr triple lumen CVP catheter (Arrow International Inc, Reading, PA, USA) was inserted into the right internal jugular vein. A pulmonary artery catheter (OPTIQ SVO2/CCO; Abbott Laboratories, North Chicago, IL, USA) was also inserted into the right internal jugular vein. Intravascular electrocardiography was used to confirm the placement of the CVP catheter in the lower superior vena cava directly above the junction of superior vena cava with the right atrium. The pulmonary artery catheter (PAC) was positioned using wedge pressure and confirmed with fluoroscopy.

Liver transplantation was performed with preservation of the retrohepatic caval vein (the piggyback technique). Blood samples were obtained simultaneously from the pulmonary artery catheter and central venous catheter at nine specific times during the procedure: Tl, immediately after insertion of the PAC (baseline); T2, 2 h after surgical incision; T3, at the end of the dissection phase; T4, at the onset of anhepatic phase (cross-clamp of portal and hepatic veins); T5, 30 min after start of the anhepatic phase; T6, end of anhepatic phase; T7, 10 min after unclamping; T8, 1 h after unclamping; and T9, end of surgery. All samples from the pulmonary artery catheter were withdrawn over 30 s using a low-negative pressure technique and never with the balloon inflated. A standard volume of 1.5 ml of blood was obtained from each site after withdrawal of dead space blood and flushing fluid. All measurements were made using a co-oximeter (ABL 700; Radiometer, Copenhagen, Denmark) calibrated daily. Immediately after a series of blood samples was drawn, mean arterial blood pressure (MAP), heart rate, and cardiac output (CO) were recorded. Cardiac output was determined by the thermodilution technique using the pulmonary artery catheter.

Data are described as mean (SD). Repeated measures ANOVA was used for comparison of cardiac output, mixed and central venous oxygen saturation with Tukey post hoc test if statistical significance was reached. The difference between mixed and central values in each individual was calculated at each time point. Comparison of the differences between the different time points was done using paired *t*-tests. The systematic error (bias) and the variability (SD of the bias) for Svo_2 vs $Scvo_2$ were calculated. Ninety-five per cent limits of agreement were calculated as bias −1.96 SD. The differences between individual values were plotted against their means for each time point (Bland–Altman plot) [11]. Correlations between variables were performed using the Pearson moment correlation equation. A p value < 0.05 was considered significant. All statistical calculations were performed using MICROSOFT EXCEL version 7 (Microsoft Corporation, Redmond, WA, USA) and SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) programs.

Results

Fifty patients (40 males and 10 females) were enrolled in the study. Their mean (SD) age was 48.3 (7.8) years, weight 78.8 (10.8) kg, and height 169 (7) cm. Forty-five patients had end-stage liver disease following hepatitis C (Child class C), three patients had auto-immune hepatitis (Child C), and two patients had hepatocellular carcinoma (Child B).

Mean values of haemodynamic parameters at the different stages are given in Table 1. Cardiac output decreased during the anhepatic phase compared with the pre-anhepatic phase and increased in the postanhepatic phase compared with both the pre- and anhepatic phases. Mean central and mixed venous oxygen saturation did not differ during T1 and T2. However, the difference between these values increased starting from T3 (see Fig. 1). The mean difference between Svo_2 and $Scvo_2$ (bias) and 95% limit of agreement are given in Table 2.

Table 1 Haemodynamic parameters during liver transplantation. Data are mean (SD).

	Pre-anhepatic phase	Anhepatic phase	Postanhepatic phase
Heart rate	91.7 (9.3)	114.7 (7.8)	108 (5.7)
Means arterial pressure; mmHg	69.7 (6.8)	56.7 (4.4)	60.3 (5.2)
Cardiac output; $l.min^{-1}$	9.5 (1.9)	8.5 (2.0)*	11.0 (2.3)†

*p < 0.05 vs pre-anhepatic phase.
†p < 0.05 vs pre-anhepatic and anhepatic phases.

© 2009 The Authors
Journal compilation © 2009 The Association of Anaesthetists of Great Britain and Ireland

13652044, 2009, 4, Downloaded from https://associationofanaesthetists-publications.onlinelibrary.wiley.com/doi/10.1111/j.1365-2044.2008.05793.x, Wiley Online Library on [09/08/2026]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

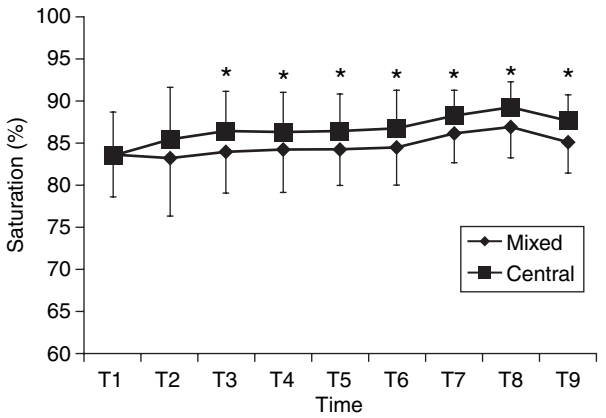

Figure 1 Mixed and central venous oxygen saturation during liver transplantion. Data are mean (SD). *$p < 0.05$. For explanation of time points T1–T9 see text.

The difference between Svo_2 and $Scvo_2$ (bias) was least at T1 (−1.94 to 2.7%), and increased from T2 up to T9 ($p < 0.05$). Figure 2(a–c) shows the Bland–Altman plot for Svo_2 vs $Scvo_2$ during the three phases of surgery. There was strong positive correlation for Svo_2 with $Scvo_2$ ($r = 0.98$ and 0.87 at T1 and T9, respectively). The correlation of venous saturations (mixed and central) with cardiac output was weak ($r = 0.35$ and 0.36 for Svo_2 and $Scvo_2$, respectively).

Discussion

Our results show that there is a good correlation between the changes in Svo_2 and $Scvo_2$ during various stages of living related liver transplantation, with a higher $Scvo_2$ than Svo_2 during most of the procedure. The mean difference of Svo_2 and $Scvo_2$ at T1 was lower compared with later time points. This can be explained by the fact that the effect of the inhalational anaesthetic had not reached steady state.

Differences between mixed and central venous saturations can vary greatly according to the pathophysiological status. Two previous animal studies have demonstrated higher $Scvo_2$ measurements compared with Svo_2 [12, 13]. Clinical trials have demonstrated that $Scvo_2$ can consistently over-estimate Svo_2 in critically ill patients and that such variation is not seen in healthy individuals [14, 15]. The hyperkinetic status associated with liver disease in our patients may be responsible for the variations we detected between mixed and central venous saturation. Mixed venous oxygen saturation is normally elevated in patients with end-stage liver disease. Arterio-venous shunting and impairment of peripheral oxygen extraction account for the hyperkinetic state usually observed in these patients [16–18].

Chawla et al. [19] showed in a mixed group of 53 critically ill patients that Svo_2 was consistently lower than $Scvo_2$ with a mean (SD) bias of −5.2 (5.1) %. They concluded that measurements of $Scvo_2$ and Svo_2 were not equivalent and concluded that $Scvo_2$ was not a reliable surrogate for Svo_2 in critically ill medical or surgical patients. Dueck et al. [7] studied 70 neurosurgical patients and made simultaneous measurements of Svo_2 and $Scvo_2$ at induction, while the patient was in the sitting position and at the end of surgery. The 95% limit of agreement ranged from −8.3% to 9.4% at induction to −6.6% to 10.0% at the end of surgery. They concluded that trends, but not individual values, of $Scvo_2$ can be used as a clinical guide to therapeutic intervention. The 95% limit of agreement of our study ranged from −1.94 to 2.7 at T1 to −6.07 to 1.07 at T9. This narrow range of difference compared with Dueck and colleagues can be attributed to the haemodynamic alterations faced during their patients' positioning.

Logically the value of Svo_2 and or $Scvo_2$ should change during the dissection phase and anhepatic phase due to the decreased cardiac output associated with clamping of the major vessels. However this did not occur in our study and Svo_2 and $Scvo_2$ remained unchanged during the three phases. Similarly, Jugan et al. [20] found that Svo_2 remained stable during the dissection and anhepatic phases. Moreover, the changes in Svo_2 expected to occur during the neo-hepatic phase was not seen since the cardiac output was extremely high in all patients. Acosta et al. [21] studied 23 cirrhotic patients undergoing liver

Table 2 Difference (bias) between mixed venous (Svo_2) and central venous ($Scvo_2$) oxygen saturation. Data are differences in mean (95% CI) and 95% limit of agreement. For explanation of time points T1–T9 see text.

	Pre-anhepatic			Anhepatic			Postanhepatic		
	T1	T2	T3	T4	T5	T6	T7	T8	T9
Svo_2 and or $Scvo_2$	0.398 (0.06 to 0.73)	−2.41 (−2.9 to −1.8)	−2.52 (−2.9 to −2.0)	−2.10 (−2.4 to −1.7)	−2.18 (−2.58 to −1.77)	−2.38 (−2.78 to −1.99)	−2.07 (−2.42 to 1.73)	−2.35 (−2.85 to −1.85)	−2.50 (−3.02 to −1.98)
95% limit of agreement	−1.94 to 2.7	−5.9 to 1.1	−5.5 to 0.4	−4.2 to 0.07	−4.9 to 0.6	−5.12 to 0.358	−4.47 to 0.326	−5.81 to 1.11	−6.07 to 1.07

© 2009 The Authors
Journal compilation © 2009 The Association of Anaesthetists of Great Britain and Ireland

13652044, 2009, 4, Downloaded from https://associationofanaesthetists-publications.onlinelibrary.wiley.com/doi/10.1111/j.1365-2044.2008.05793.x, Wiley Online Library on [09/08/2026]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

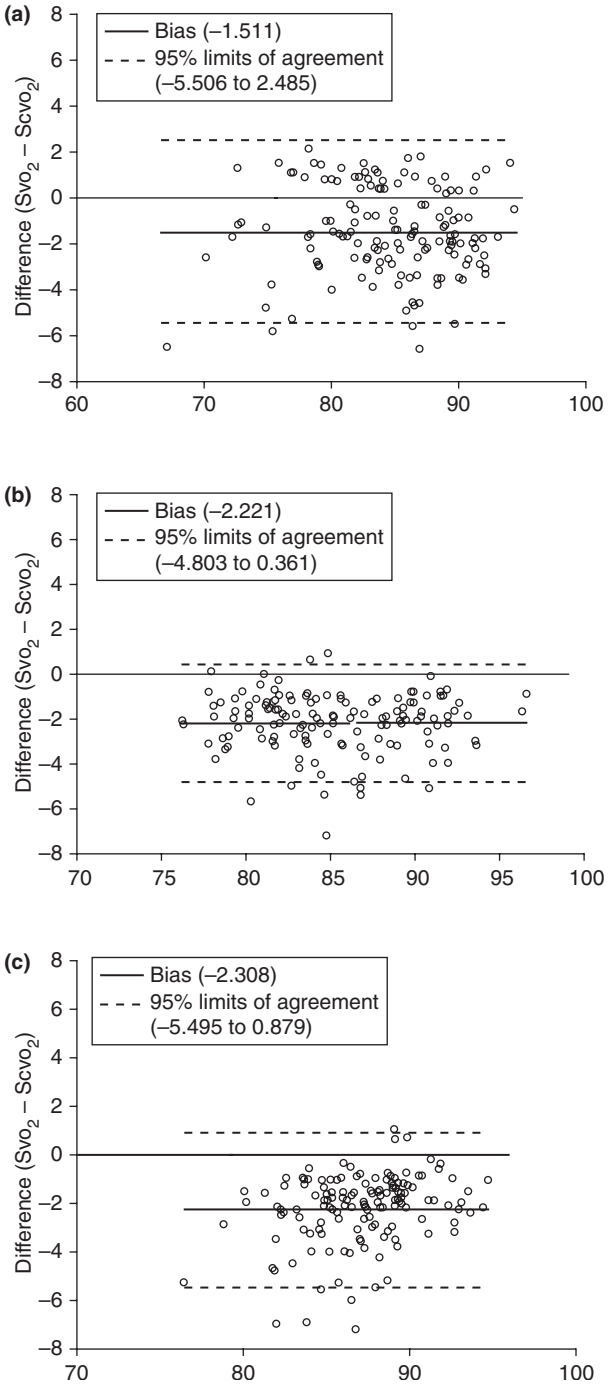

Figure 2 Bland–Altman plots of the differences between mixed venous oxygen saturation (Svo_2) and central venous oxygen saturation ($Scvo_2$) during the pre-anhepatic phase (a), anhepatic phase (b) and postanhepatic phase (c) of liver transplantation. The unbroken line indicates the mean difference (bias), and the broken lines indicate the 95% limits of agreement.

transplantation using the piggyback technique and demonstrated that Svo_2 changes were minimal when the cardiac index exceeds 4 $l.min^{-1}.m^{-2}$. In our study the mean cardiac output ranged from 8.5 to 11.4 $l.min^{-1}$. In our study the mean Svo_2 was approximately 80% throughout and showed a poor correlation with cardiac output. Jugan et al. [20] also reported that the correlation between Svo_2 and cardiac output was poor.

One limitation of the current study is that the studied population normally exhibits a hyperdynamic status resembling patients with sepsis, although pathophysiological state is different. Our finding cannot be extrapolated to patients with sepsis or patients with low or moderate cardiac output. Another limitation is that the huge haemodynamic changes that occur intra-operatively in patients undergoing liver transplantation are expected and rapidly managed, thus minimising any sustained haemodynamic variation with subsequent reflection in venous oxygen saturation. The high values of Svo_2 and $Scvo_2$ in this study does not reflect major changes in oxygen supply/demand relationship that appear if Svo_2 and $Scvo_2$ are greatly decreased (60–70%). The Svo_2 and $Scvo_2$ were consistently greater than the reference range throughout the study period and we cannot state with confidence that agreement between Svo_2 and $Scvo_2$ exists at lower values.

In conclusion, we found a small bias between central and mixed venous oxygen saturation in patients undergoing liver transplantation with a hyperdynamic cardiovascular system and that central venous oxygen saturation may be used as a surrogate for mixed venous oxygen saturation.

References

1 Kandel G, Aberman A. Mixed venous oxygen saturation. Its role in the assessment of the critically ill patient. *Archives of Internal Medicine* 1983; **143**: 1400–2.
2 Miller MJ. Tissue oxygenation in clinical medicine: an historical review. *Anesthesia & Analgesia* 1982; **61**: 527–35.
3 Reinhart K, Bloos F. The value of venous oximetry. *Current Opinion in Critical Care* 2005; **11**: 259–63.
4 Edwards JD, Mayall RM. Importance of the sampling site for measurement of mixed venous oxygen saturation in shock. *Critical Care Medicine* 1998; **26**: 1356–60.
5 Martin C, Auffray JP, Badetti C, Perrin G, Papazian L, Gouin F. Monitoring of central venous oxygen saturation versus mixed venous oxygen saturation in critically ill patients. *Intensive Care Medicine* 1992; **18**: 101–4.
6 Rigg JD, Nightingale PN, Faragher EB. Influence of cardiac output on the correlation between mixed venous and central venous oxygen saturation. *British Journal of Anaesthesia* 1993; **71**: 459.
7 Dueck MH, Klimek M, Appenrodt S, Weigand C, Boerner U. Trends but not individual values of central venous oxygen saturation agree with mixed venous oxygen saturation during varying hemodynamic conditions. *Anesthesiology* 2005; **103**: 249–57.

© 2009 The Authors
Journal compilation © 2009 The Association of Anaesthetists of Great Britain and Ireland

13652044, 2009, 4, Downloaded from https://associationofanaesthetists-publications.onlinelibrary.wiley.com/doi/10.1111/j.1365-2044.2008.05793.x, Wiley Online Library on [09/08/2026]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

8 Bechstein WO, Neuhaus P. [Bleeding problems in liver surgery and liver transplantation]. *Chirurg* 2000; **71**: 363–8.
9 Rossi G, Langer M, Maggi U, et al. Veno-venous bypass versus no bypass in orthotopic liver transplantation: hemodynamic, metabolic, and renal data. *Transplantation Proceedings* 1998; **30**: 1871–3.
10 Bulkley GB. Reactive oxygen metabolites and reperfusion injury: aberrant triggering of reticuloendothelial function. *Lancet* 1994; **344**: 934–6.
11 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; **1**: 307–10.
12 Reinhart K, Rudolph T, Bredle DL, Hannemann L, Cain SM. Comparison of central-venous to mixed-venous oxygen saturation during changes in oxygen supply/demand. *Chest* 1989; **95**: 1216–21.
13 Schou H, Perez de Sa V, Larsson A. Central and mixed venous blood oxygen correlate well during acute normovolemic hemodilution in anesthetized pigs. *Acta Anaesthesiologica Scandinavica* 1998; **42**: 172–7.
14 Reinhart K, Kuhn HJ, Hartog C, Bredle DL. Continuous central venous and pulmonary artery oxygen saturation monitoring in the critically ill. *Intensive Care Medicine* 2004; **30**: 1572–8.
15 Scheinman MM, Brown MA, Rapaport E. Critical assessment of use of central venous oxygen saturation as a mirror of mixed venous oxygen in severely ill cardiac patients. *Circulation* 1969; **40**: 165–72.
16 Birman H, Haq A, Hew E, Aberman A. Continuous monitoring of mixed venous oxygen saturation in hemodynamically unstable patients. *Chest* 1984; **86**: 753–6.
17 Viale JP, Annat G, Delafosse B, Bouffard Y, Tissot S, Motin J. Measurement of gas exchange in anesthesia and during resuscitation: principles and applications. *Agressologie* 1990; **31**: 62–3.
18 Waller JL, Kaplan JA, Bauman DI, Craver JM. Clinical evaluation of a new fiberoptic catheter oximeter during cardiac surgery. *Anesthesia & Analgesia* 1982; **61**: 676–9.
19 Chawla LS, Zia H, Gutierrez G, Katz NM, Seneff MG, Shah M. Lack of equivalence between central and mixed venous oxygen saturation. *Chest* 2004; **126**: 1891–6.
20 Jugan E, Albaladejo P, Jayais P, Descorps-Declere A, Ecoffey C. Continuous monitoring of mixed venous oxygen saturation during orthotopic liver transplantation. *Journal of Cardiothoracic and Vascular Anesthesia* 1992; **6**: 283–6.
21 Acosta F, Sansano T, Palenciano CG, et al. Does mixed venous oxygen saturation reflect the changes in cardiac output during liver transplantation? *Transplantation Proceedings* 2002; **34**: 277.

© 2009 The Authors
Journal compilation © 2009 The Association of Anaesthetists of Great Britain and Ireland

13652044, 2009, 4, Downloaded from https://associationofanaesthetists-publications.onlinelibrary.wiley.com/doi/10.1111/j.1365-2044.2008.05793.x, Wiley Online Library on [09/08/2026]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License